Case Report

Restoration of Consciousness with Zolpidem in a Patient with Sporadic Creutzfeldt - Jakob Disease (sCJD)

Roshan Sutar1*, Abhinav Nahar1, Seby Kurthukulangara1, Ravinder Jeet Singh Sidhu2, Harish T1, Jitender Saini3, Ravi Yadav1 and Santosh Chaturvedi1

1Department of Psychiatry, National Institute of Mental Health and Neurosciences, Bangalore, India
2Department of Neurology, National Institute of Mental Health and Neurosciences, Bangalore, India
3Department of Neuroradiology, National Institute of Mental Health and Neurosciences, Bangalore, India

Abstract

Creutzfeldt-Jakob Disease (CJD) is a rare form of dementia with rapid progression and spongiform degeneration of brain secondary to accumulation of misfolded prion proteins. Sporadic CJD presents with rapidly progressing cognitive decline, myoclonus, motor and behavioural disturbances. As the disease advances, consciousness is progressively impaired; different pharmacological agents and deep brain stimulation have been tried to improve consciousness with minimal evidence. Zolpidem is a non-benzodiazepine hypnotic agent and also has an arousal action and has been reported as an "awakening drug" in some patients with disorders of consciousness. We report a patient with sCJD whose consciousness markedly improved with a zolpidem.

Keywords: Consciousness; Creutzfeldt - Jakob disease; Zolpidem

Introduction

Creutzfeldt - Jakob disease is a rare form of dementia associated with neuropathological finding of accumulation of prion proteins in the brain with rapid deterioration and disability [1]. Alteration in the level of consciousness has been studied in dementia and other hypoxic brain injuries. Attempt to modulate the effect of pharmacological agents and deep brain stimulation to attain the maximum level of consciousness is ongoing but the evidence is lacking [2]. This report supports the use of zolpidem in the arousal from altered consciousness in a patient with sporadic CJD.

*Corresponding author: Roshan Sutar, Department of Psychiatry, National Institute of Mental Health and Neurosciences, Bangalore, India, Tel: +91 9980590172; E-mail: roshidoc@yahoo.co.in

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Discussion

In our patient, clinical, electrophysiological and radiological features supported diagnosis of sCJD, which is thought to have an irreversible, rapidly progressive course [3]. Deterioration in the levels of consciousness during disease progression was a prominent feature in our patient [4].
Zolpidem preferentially acts on omega 1-GABA-A receptors within the GABA receptor - chloride channel complex. Arousal appears to be specific to zolpidem unlike other non-specific benzodiazepines due to possible differential receptor affinity, but exact mechanism is still unclear. One hypothesis is that reduced arousal in patients following brain disease leads to alteration in the number of GABA receptors as a mechanism of neuroprotection. Zolpidem appears to reverse it by reactivation of dormant brain neural networks. Another hypothesis claims that zolpidem may activate limbic loops modulating motivational processes thus leading to disinhibition and thalamo-cortical overactivity [5]. Paradoxical restoration of consciousness by CNS depressants is thought to be by functional restoration of certain networks like reticular activating system, cortico-cortical loop and cortico-thalamo-cortical loop as suggested by functional neuroimaging studies [3,6,7]. However, not all patients with disorders of consciousness respond to zolpidem as noted in a placebo controlled trial [2].

In conclusion, zolpidem appears to have a role in improving consciousness in some patients with disorders of consciousness such as CJD. Though response is likely to be only for a short period, the very fact that improvement albeit transient is possible, in rapidly progressive neurodegenerative diseases otherwise deemed to be irreversible, merits further studies which may also enhance our understanding of neurobiology of consciousness [7].

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Conflict of Interest

None

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